anonymous

Bio 222

Annotated Bibliography

2/19/07

The genetic bottleneck of the African cheetah: its effect on their genetic variation and future survival.

Amos W and Harwood J. 1998. Factors affecting levels of genetic diversity in natural populations. Philos.Trans.R.Soc.Lond.B.Biol.Sci. 353(1366):177-86.

The two authors of this review article work for the Department of Zoology and the University of St. Andrews, both in the UK. It was published nine years ago and appears to be intended for a more novice scientific audience interested in factors contributing to genetic diversity. This article is important to my topic because it provides a basis by which genetic diversity is affected, other than bottlenecks. The majority of my other research articles focus solely on the genetics of the bottleneck. Several factors that affect diversity and are listed in this paper are inbreeding depression and heterozygote instability. Inbreeding depression is the decrease in fitness through the increased expression of deleterious (harmful) alleles; it is much more common in small population sizes. Heterozygote instability proposes that mutation will increase with heterozygosity, therefore boosting genetic variation in already diverse populations.

De Smet K. 1993. Cheetahs teetered on brink in the ice age. New Sci. 138(1875):16.

The author of this article works for New Scientist magazine and reviews the work of O'Brien and Menotti-Raymond (below). It is not as complex as others I have read and is intended for anyone interested the nature of the cheetahs' genetic bottleneck. O'Brien and Menotti-Raymond analyzed mitochondrial DNA because of its high rate of mutation. They cleaved mtDNA with specific restriction enzymes, looking for particular size fragments. When a fragment of a different size was found, they knew a mutation had occurred. Their results support a genetic bottleneck of around 10000 years ago, a time period when seventy-five percent of all large mammals died out. Even more intriguing is that all present-day cheetahs could have descended from only one to two litters.

Drake GJ, Kennedy LJ, Auty HK, Ryvar R, Ollier WE, Kitchener AC, Freeman AR, Radford AD. 2004. The use of reference strand-mediated conformational analysis for the study of cheetah (acinonyx jubatus) feline leukocyte antigen class II DRB polymorphisms. Mol.Ecol. 13(1):221-9.

The authors of this article work for the University of Liverpool, University of Manchester, and the National Museums of Scotland. The intended audiences of this piece are scientists interested in the genetic variation of cheetahs and in alternative ways to study their variation. The major histocompatibility complex (MHC) is a very common region used to analyze genetic variability, however, this study uses reference strand-mediated conformational analysis to evaluate genetic variation in cheetahs by examining the feline leukocyte antigen (FLA). Extending from genetic variability, the authors also address the disease susceptibility due to the cheetahs' high monomorphism. A very useful graph is presented to show the degree of variation in the FLA region.

Menotti-Raymond M and O'Brien SJ. 1993. Dating the genetic bottleneck of the african cheetah. Proc.Natl.Acad.Sci.U.S.A. 90(8):3172-6.

The authors of this article work for branches of the National Cancer Institute, Frederick, MD. It appears to be intended for scientists interested in the general time period when the cheetah underwent a genetic bottleneck. Many studies have been conducted to show the low genetic variability in the cheetah, but this appears to be a pioneer article in dating their bottleneck. To estimate the time of the bottleneck, scientists examined mtDNA, which evolves five to ten times faster than nuclear DNA in mammals. Mitochondrial DNA is very useful in examining bottlenecks because of its recombinant free pattern of maternal inheritance, which increases its sensitivity to demographic disruptions. The results support a bottleneck in the cheetah population 6000-20000 years ago.

Munson L, Terio KA, Worley M, Jago M, Bagot-Smith A, Marker L. 2005. Extrinsic factors significantly affect patterns of disease in free-ranging and captive cheetah (acinonyx jubatus) populations. J.Wildl.Dis. 41(3):542-8.

The authors of this article work for University of California, Zoological Society of San Diego, Otjiwarango Veterinary Clinic, and the Cheetah Conservation Fund. This primary article is intended for scientists interested in how captive and wild cheetahs differ in health, even though they are genetically similar. The authors acknowledged the lack of polymorphism in the MHC region and proposed that captive and wild cheetahs would have comparable health issues. The data ended up in support of the opposite; wild cheetahs were in much better health than captive cheetahs. This data infers that cheetahs, because of their high monomorphism, have a hard time adapting to environmental change (i.e. going from the wild to captivity). Also, other factors than genetics alone play a large part in the cheetahs' health. With this information, the authors conclude that the emphasis should be placed on the conservation of the cheetahs' homeland, instead of capture and reintroduction to the wild.

. O'Brien SJ, Roelke ME, Marker L, Newman A, Winkler CA, Meltzer D, Colly L, Evermann JF, Bush M, Wildt DE. 1985. Genetic basis for species vulnerability in the cheetah. Science 227(4693):1428-34.

This review article was published in Science magazine; the authors' line of work was not listed. The intended audiences of this article are people with at least some scientific

background interested in cheetahs and their future. Like many other articles, this reviews the MHC for its genetic uniformity in the cheetah. However, once the monomorphism of the cheetah was recognized, implications resulting from their uniformity are discussed. These implications include high juvenile mortality, difficulty of captive breeding, and high susceptibility to several pathogens. This is important to my research because it expounds on the genetic uniformity of the cheetah, noting several more important factors directly related to their bottleneck.

Penn D. 2002. Major histocompatibility complex. Leishman WB, editor. In: Encyclopedia of life sciences. London: Nature Publishing Group. 338 p.

The author of this article works for the University of Utah. This reference source appears to be a fairly advanced piece and is intended for people wanting to know about the major histocompatibility complex (MHC). Much of my other research studies the MHC and how it can be analyzed to detect for genetic variability; they assume you have some knowledge about the MHC. This article, however, focuses solely on the MHC and its function/genetic makeup. The MHC is a large region of DNA that makes up about .1% of the human genome and is the most polymorphic region in vertebrates. The MHC genes encode cell surface receptors which aid in self and non-self recognition. This ultimately has consequences dealing with tissue rejection, autoimmunity, and response to pathogens.

Yuhki N and O'Brien SJ. 1990. DNA variation of the mammalian major histocompatibility complex reflects genomic diversity and population history. Proc.Natl.Acad.Sci.U.S.A. 87(2):836-40.

The authors of this article work for the Laboratory of Viral Carcinogenesis at the National Cancer Institute in Frederick, Maryland. Its intended audience is for scientists interested in the genetic variability observed in the African cheetah and the Asiatic lion. The genetic variability was determined by studying the major histocompatibility complex (MHC). The MHC is a complex of genes that play an important role in immune regulation and response to foreign antigens. High variability in the MHC is preferred because the immune system would be able to defend against numerable pathogens. Low variability would provide solid defense against a few pathogens, but little defense when in the presence of others. The authors' study concludes an extremely low variation in the MHC of the African cheetah, which may have implications on their future survivability.